#### Annals of Clinical and Analytical Medicine

**Original Research** 

# Do body composition, hemogram and lipids differ between obese women with and without gonarthrosis?

Gonarthrosis and body composition

Gökhan Peker<sup>1</sup>, Dila Mete Peker<sup>2</sup>, Rahman Köseoğlu<sup>3</sup>, Orkun Gül<sup>4</sup> <sup>1</sup> Department of Orthopaedics And Traumatology, University of Health Sciences, Trabzon Kanuni Education and Research Hospital <sup>2</sup> Department of Internal Medicine, Trabzon Kanuni Education and Research Hospital <sup>3</sup> Department of Internal Medicine, Trabzon Yıldızlı Medicalpark Hospital <sup>4</sup> Department of Orthopaedics And Traumatology, Trabzon Yıldızlı Medicalpark Hospital, Trabzon, Turkey

This study was presented at "Bone Joint Congress" as oral presentation, Girne, Northern Cyprus, 2022

#### Abstract

Aim: Knee osteoarthritis (KOA) is due to fat mass (FM) rather than lean mass (LM) in the body. In this study, we aimed to investigate differences in body composition in obese women with and without gonarthrosis; we also aimed to investigate serum inflammatory and lipid values between the groups. Material and Methods: Ninety-six women were separated into gonarthrosis and control groups and retrospectively analyzed. Body composition analysis was performed by the TANITA device. The neutrophil/lymphocyte (NLR) and monocyte/lymphocyte (MLR) ratios, low-density (LDL-C) and high-density lipoprotein

cholesterol (HDL-C) values, triglyceride (TG) values, and TG/HDL-C ratio of the two groups were compared. Results: Body mass index, body and leg fat ratio, body fat mass (BFM) to body lean mass (BLM) ratio, leg fat mass (LFM) to leg lean mass (LLM) ratio were higher in the gonarthrosis group (p<0.001). BLM and LLM percent was higher in the control group (p<0.001). There was no significant difference in serum

inflammatory and lipid values except for HDL-C. Discussion: Both groups consisted of obese women. It was observed that while the rate of BFM and LFM was high in people with gonarthrosis, the rate of LM was low. This is the first study comparing the body composition of obese patients with and without gonarthrosis. The primary implication in this study is that weight reduction in individuals with obesity and gonarthrosis should primarily target adipose tissue.

#### Keywords

Gonarthrosis, Body Composition, Obesity, Women, Bioelectrical Impedance Analysis

DOI: 10.4328/ACAM.21540 Received: 2022-12-06 Accepted: 2023-01-20 Published Online: 2023-02-04 Printed: 2023-05-01 Ann Clin Anal Med 2023;14(5):399-403 Corresponding Author: Gökhan Peker, Department Of Orthopaedics And Traumatology, Trabzon Kanuni Education and Research Hospital, 61250, Yomra, Trabzon, Turkey. E-mail: drgokhanpeker@gmail.com P: +90 532 588 70 51

Corresponding Author ORCID ID: https://orcid.org/0000-0002-6211-6645

This study was approved by the Ethics Committee of University of Health Sciences, Trabzon Kanuni Education and Research Hospital (Date: 2020-12-23, No: 2020/84)

### Introduction

Although the incidence and treatment costs of knee osteoarthritis (KOA) are so high, however, ideal treatment methods for gonarthrosis have not been found yet [1]. Therefore, it is important to prevent causative factors. Age, gender, body weight, impaired lipid metabolism, adipose tissue inflammation and joint trauma may all play a role [2, 3].

The human body is composed of fatty mass (FM) and lean mass (LM) that includes muscle and bone, which can not be determined using body mass index (BMI) [3].

In the pathogenesis of KOA, the immune system is activated [4]. Neutrophil-to-lymphocyte ratio (NLR) and monocyte-to-lymphocyte ratio (MLR) may reflect the balance of the immune response [4].

We posited that persons with obesity and KOA may have dyslipidemia, higher inflammatory values like MLR and NLR, higher percentage of body fat mass (BFM) and higher leg fat mass (LFM), not lean mass (LM), than individuals without KOA. This study tested this hypothesis by identifying significant differences in body composition, serum lipid values, and complete blood count parameters comparing obese women with and without gonarthrosis.

### Material and Methods

The study was a retrospective case-control study and was approved by the local Ethics Committee of the authors' institution. Two hundred thirty-five patients admitted to the obesity center of the hospital between 2019 and 2020 were retrospectively investigated for the study. Informed consent was obtained from all participants.

Hemogram and lipid tests were routinely performed at the time of admission. Patients were classified according to the World Health Organization classification for obesity (Table 1).

Body composition parameters, including FM (kg) and LM (kg), were measured by the TANITA MC 780 MA segmental body composition analyzer, consisting of a weighing platform that performs bioelectrical impedance analysis (BIA) of a barefooted standing person. Body composition differs by gender, with men having more LM and women having more FM [5]; thus the study included only women to eliminate gender differences. Ninety-six of 235 patients who had sufficient data on retrospective examination were included in the study. The body composition analyzer calculated body fat mass (BFM), leg fat mass (LFM), body lean mass (BLM), leg lean mass (LLM), body skeletal muscle mass (BSM), leg skeletal muscle mass (LSM) and percentages of FM and LM. These results were proportioned according to the load-capacity model to obtain BFM/BLM and LFM/LLM results [6]. Percentages (%) of BFM, LFM, BLM, LLM, LFM/BFM and LSM/BSM were calculated. The leg muscle mass index (LMMI) was established to measure relative muscle mass relative to body size by dividing leg muscle mass (kg) by the square of body length (m2) [7].

Patients with systemic pathology, including any inflammatory joint disease, infection, any chronic renal failure, a history of musculoskeletal trauma or surgery, and those who received corticosteroids, anti-inflammatory and lipid-lowering drugs were excluded from the study.

Patients were divided into two groups. Forty-eight obese

patients who had stage 2 and higher osteoarthritis were included in the gonarthrosis group. Forty-eight patients with obesity without joint pain complaints and normal knee radiographs were included in the control group. In determining the stage of osteoarthritis, the Kellgren-Lawrence classification, which is the WHO standard for epidemiological studies, was used [8]. Patient radiographs were separately evaluated by two different orthopedists, each unaware of the other. In cases of conflict, a third was consulted.

Based on laboratory tests, the NLR, MLR, LDL-C, HDL-C, triglyceride (TG), TG/ HDL-C values were compared between the two groups.

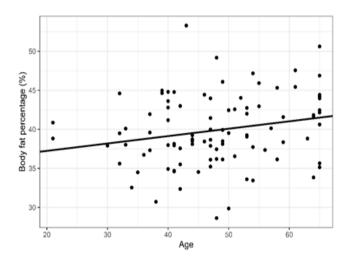
The mean differences between the control (n = 48) and gonarthrosis (n = 48) groups were compared for the variables of interest. Before the comparison, the normality of groups was assessed with the Shapiro-Wilk test, and an independent sample t-test was used to compare the means for variables that were determined to be normally distributed (p<0.05, for Shapiro-Wilk). Afterward, the remaining variables (p>0.05, for Shapiro-Wilk) were tested with a non-parametric Mann-Whitney U test. Different stages of gonarthrosis, which are classified as control group (n=48), stage 2 (n=16), stage 3 (n=12), and stage 4 (n=20) were also compared for the same interested variables. Due to the small stage 3 sample size, these comparisons were used with the Kruskal-Wallis test. Finally, Dunn's post hoc procedure was used for the pairwise comparison of each gonarthrosis stage using adjusted p-values. P-values less than 0.05 were considered statistically significant. The analysis was conducted in SPSS version 26.0, and figures were produced in an R package ggplot2.

### Ethical Approval

Ethics Committee approval for the study was obtained.

#### Results

One hundred ninety-two legs of 96 patients were examined. In the gonarthrosis group, there were 16 patients with stage 2, 12 patients with stage 3 and 20 patients with stage 4 gonarthrosis patients. Gonarthrosis was bilateral in all patients. There was no difference in composition between the two legs in any of the patients. Each patient's leg was randomly selected for composition analysis. The average age in the control group





### Table 1. WHO classification of obesity

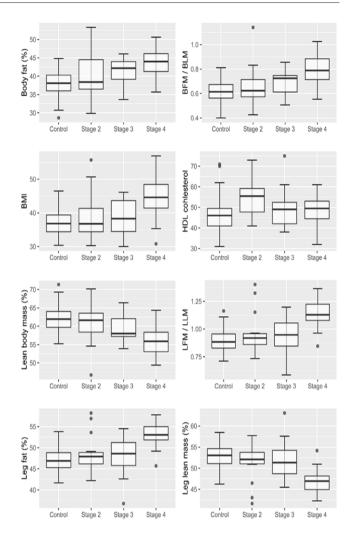
Classification	WHO criteria BMI cut-off (kg/m²)
Underweight	<18.5
Normal	18.5-24.9
Overweight	≥25
Pre-obese	25.0-29.9
Obese	≥30
Obese class 1	30-34.9
Obese class <sup>2</sup>	35-39.9
Obese class <sup>3</sup>	≥40

**Table 2.** Comparisions between gonarthrosis stages andcontrol groups

Variables	Control	2. Stage	3. Stage	4. Stage	⁻ p-value	
BFM%	38.07	38.38	42.15	43.97	<0.01	
BLM%	61.93	61.62	58.00	55.91	<0.01	
LLM%	53.30	52.23	51.55	47.27	<0.01	
LFM %	46.65	47.85	48.50	52.70	<0.01	
LFM / BFM	0.22	0.23	0.22	0.23	0.324	
LSM / BSM	0.16	0.16	0.15	0.16	0.319	
BFM/ BLM	0.61	0.62	0.72	0.79	<0.01	
LFM / LLM	0.88	0.91	0.94	1.12	<0.01	
LSM (kg) / height <sup>2</sup> (m <sup>2</sup> )	3.36	3.44	3.32	3.69	0.068	
BMI	36.80	36.75	38.30	44.60	<0.01	
LDL	128.50	151.50	126.00	141.00	0.114	
HDL	46.00	55.50	49.00	49.50	<0.01	
Neutrophils / Lymphocytes	1.50	1.27	1.81	1.47	0.063	
Tg	102.50	126.50	116.00	130.50	0.611	
Tg/Hdl	2.27	2.48	2.76	2.52	0.671	
Monocytes / Lymphocytes	0.20	0.19	0.19	0.20	0.430	

The Kruskal-Wallis test results for variables (LDL: low-density ignificant, HDL: high--density lipoprotein, BFM%: Body fat mass percentage, LFM%: leg fat mass percentage, BLM%: body lean mass percentage, LLM% Leg lean mass percentage, LSM: leg straight muscle mass, BSM: body straight muscle mass)

## Table 3. Comparison of control and gonarthrosis groups



**Figure 2.** Box plots for variables with at least one significant difference between groups with the Kruskal-Wallis test (HDL: high-density lipoprotein, BMI: body mass index, BFM%: Body fat mass percentage, LFM%: leg fat mass percentage, BLM%: body lean mass percentage, LLM%: Leg lean mass percentage.)

	<b>A</b>		e				
	Average		Standard deviation		Standard error (for M)		- p-value
	Gonarthrosis	Control	Gonarthrosis	Control	Gonarthrosis	Control	p vulue
BMI	41.363	37.358	6.849	4.334	0.999	0.626	<0.001
BFM%	41.740	38.089	4.764	3.601	0.695	0.520	<0.001
LFM%	50.294	46.905	4.522	2.951	0.660	0.426	< 0.001
BLM%	58.310	61.891	4.862	3.607	0.709	0.521	<0.001
LLM%	49.698	53.064	4.523	2.955	0.660	0.427	< 0.001
BFM/ BLM	0.727	0.621	0.147	0.094	0.021	0.014	<0.001
LFM / LLM	1.028	0.890	0.180	0.106	0.026	0.015	< 0.001
LSMI [(LSM weight (kg) / body height <sup>2</sup> (m <sup>2</sup> )]	3.557	3.413	0.483	0.364	0.071	0.053	0.106
LSM / BSM	0.158	0.156	0.010	0.007	0.002	0.001	0.230
LFM / BFM	0.227	0.227	0.023	0.017	0.003	0.002	0.927
Neutrophils / Lymphocytes	1.619	1.6163	0.681	0.501	0.099	0.072	0.983
Monocytes / Lymphocytes	0.148	0.137	0.050	0.043	0.007	0.006	0.266
LDL	139.851	132.167	28.136	31.641	4.104	4.567	0.2144
TG	146.553	144.083	104.982	101.862	15.31	14.702	0.9076
HDL	50.6596	46.354	9.241	7.948	1.348	1.147	0.0168

Comparison of the groups in terms of laboratory findings and body composition (LDL: low-density lipoprotein, HDL: high-density lipoprotein, TG: Triglyceride, BMI: body mass index, BFM%: Body fat mass percentage, LFM%: leg fat mass percentage, BLM%: body lean mass percentage, LLM%: Leg lean mass percentage, LSM: leg straight muscle mass, LSMI: leg straight muscle mass.) (42.65  $\pm$  9.19) was less than in the gonarthrosis group (53.57  $\pm$  8.60). The correlation between age (48.05  $\pm$  10.42) and fat mass ratio (39.90  $\pm$  4.58) was evaluated for all gonarthrosis and control group subjects. Pearson's correlation test identified a weak relationship between variables (r = 0.216, p-value = 0.036). A strong relationship is not seen in the scatter plot (Figure 1).

BFM%, LFM%, BFM/BLM and LFM/LLM were significantly higher in the gonarthrosis group (p<0.001), while BLM% and LLM% were higher in the control group (p<0.001).

The Shapiro-Wilk test determined that groups of TG and TG/ HDL variables were not normally distributed (p<0.01 for each group). Thus, the Mann-Whitney U test was applied to these two variables, and no significant difference was found for these variables (p=0.228 and p=0.970, respectively).

### Comparison of disease stages

In Table 2, median values for each stage and p-values for the Kruskal-Wallis test are shown.

A significant difference was observed between the 4th stage gonarthrosis and control groups in terms of BFM% (p<0.01) and BLM% (p<0.01). A significant difference was observed between the 4th- stage and 2nd- stage gonarthrosis groups in terms of BFM% (p=0.028) and BLM% (p<0.01). A significant difference in terms of LLM% was determined between the 4th-stage group and all other groups (p<0.01 for all three). There was a significant difference in HDL cholesterol between the control group and the gonarthrosis group (p<0.01). Similarly, by examining the box graphs given in Figure 2, it can be observed that the difference generally occurs in the 4th stage (excluding HDL -C). No significant difference was found between the two groups in terms of serum LDL-C, TG values, NLR, MLR and TG-HDL-C ratio values (p>0.05), (Table 3).

Figure 2 presents comparisons between disease stages for the remaining variables.

### Discussion

This is the first study to show a difference in body composition between obese women with and without gonarthrosis. Although most previous analyses have relied on data from studies with dual-energy X-ray absorptiometry (DEXA), this study differs in collecting data obtained by BIA and using a control group consisting of women with obesity. There are many studies showing that OA patients have higher weight and BMI than those without OA, with no clear information on whether this difference is due to FM or LM. The reason of this difference may shed light on methods of reducing excess weight, which is a cause of gonarthrosis. This study demonstrates that KOA is associated with high BFM and LFM ratio in women with obesity and determines the ratio of BLM and LLM to be higher in patients with obesity without gonarthrosis (Table 3). As the stage of osteoarthritis increased, a significant increase was observed in the ratio of body and leg fat.

It has been reported that individuals of the same age, height and weight may differ in body shape, composition and energy needs. It is recognized that body composition can independently affect health [9]. One classification of abnormal body composition phenotype as a load-capacity model [load being FM (fat mass) and capacity LM (lean mass)] was calculated as the ratio of FM/

402 | Annals of Clinical and Analytical Medicine

LM [6]. The load capacity model is also a method of determining the excess fat mass (FM) as well as low lean soft tissue (LST) in people with class II/III obesity [10]. In our study, according to the load capacity model, it was observed that women with gonarthrosis had greater metabolic load than the control group (p<0.001). According to this result, gonarthrosis adds a burden to the already increased metabolic load in obesity.

There are several body composition measurement methods exist, including BIA, quantitative magnetic resonance, air displacement plethysmography, DEXA, MRI, MR spectroscopy, PET/computed tomography, and PET/MRI [9]. Our study used a BIA. BIA devices are commonly used in clinical practice and research studies [9]. Compared to MRI and DXA, BIA is fast, cheap and there is no risk of radiation exposure. Recent developments in BIA technologies involve systems that incorporate multiple frequencies (MF-BIA) and multiple body segments. The TANITA BIA system achieves a valid measurement of body fat percentage in older adults and is convenient and practical for use in public health settings [11].

A study utilizing DXA whole-body scan (Hologic QDR-4500) found that women with KOA have a greater FM than non-OA individuals. Thus, the association between BMI and OA is mainly mediated by FM [3]. Lee et al. studied the ratio of leg to whole-body muscle mass and leg to whole-body fat mass and found them to be lower and higher, respectively, in the knee OA group. They used DXA to measure leg muscle mass and found a significant association with knee pain in people with radiographic KOA (rKOA). They reported that low leg muscle mass is a useful clinical indicator of symptomatic KOA, and that DXA is a potentially excellent tool for quickly assessing leg mass in patients with KOA [12]. Abbate et al. conducted a comparative study with DXA between female patients with rKOA and those without and found significantly higher mean BMI, weight, FM, percent FM, LM in women with rKOA. Compared with women without rKOA, women with rKOA had significantly lower mean percent LM [13]. Utilizing BIA, Sowers et al. found FM, LM and straight muscle mass (SMM) to be greater in women with KOA at rates of 41%, 11.5%, 10%. Women with KOA had lower SMM to FM ratios. Mean BMI was 24% greater in women with KOA. FM and SMM were associated with K&L KOA score [14]. Our study found no significant difference between the study and control groups in terms of the leg-to-body muscle mass and leg-tobody fat mass ratios. Our study did not include clinical findings such as pain and stiffness and our study's measurements were made by the BIA method. As the ratio of BFM was high, the ratio of LM, including muscle mass, was low in osteoarthritis. Body fat was statistically significant in proportion to the stage of the disease, and an inverse relationship was found regarding the percentage of LM. While the BMI in the gonarthrosis group was significantly higher than in the control group, our study observed that it increased in parallel with the gonarthrosis stage (Table 2).

A high BMI has been associated with increased odds ratios of KOA (2.81) and hand OA (2.59), but not hip (1.11)[15]. These findings suggest that OA is not just caused by overload. Recent studies have suggested that adipose tissue inflammation and lipid metabolism disruption play important roles in obesity-related OA [16, 17]. Dyslipidemia in obesity is characterized

by high plasma levels of TGs and free fatty acids (FFAs), low levels of HDL-C with HDL dysfunction, and normal or slightly increased levels of LDL-C [18]. Disturbances in HDL metabolism, together with a different factor, can cause problems in cartilage homeostasis [16]. HDL-C has different effects, such as inhibiting inflammation in the absence of an acute phase response (APR), but increasing inflammation in the presence of an APR [19]. Although it has been reported that HDL-C values mostly decrease with inflammation and gonarthrosis, there are also publications showing the opposite [20-22]. We found HDL-C values to be higher in gonarthrosis patients in our study (p<0.05).

Adipose tissue inflammation is also seen as a characteristic feature in obesity, as cytokines, which are secreted from adipocytes and immune cells cause low-grade inflammation. In addition to systemic cytokine production, the infrapatellar fat pad produces cytokines [15]. MLR and NLR were also studied in KOA patients, who exhibited a significant increase compared to the control group. It has been reported that MLR has a high diagnostic value [4]. We also analyzed these values in our study but found no significant difference between the two groups.

### Conclusion

Our BIA-based body composition analysis of patients with obesity and knee osteoarthritis included only female patients, yielding a simpler comparison opportunity in our study. When comparing two obese groups, patients with gonarthrosis were found to have a higher percentage of BFM and LFM and lower LM. There was no significant difference between the two groups in serum inflammatory and lipid values except HDL-C. The primary implication in this study is that weight reduction in individuals with obesity and gonarthrosis should primarily target adipose tissue. Our study is the first to include persons with obesity as a control group in the investigation of body composition in osteoarthritis and we believe it will shed light on new studies. Prospective studies with larger sample sizes are needed in the future.

#### Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

#### Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

### Funding: None

#### Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

#### References

1. Erduran M, Akseki D, Karaoğlan O, Pinar H. Patellofemoral joint dynamics in patiens with gonarthrosis. Eklem Hastalik Cerrahisi. 2009; 20(1):18-24.

2. Godziuk K, Prado CM, Woodhouse LJ, Forhan M. The impact of sarcopenic obesity on knee and hip osteoarthritis: a scoping review. BMC Musculoskelet Disord. 2018; 19(1):271.

3. Ho-Pham LT, Lai TQ, Mai LD, Doan MC, Nguyen TV. Body Composition in Individuals with Asymptomatic Osteoarthritis of the Knee. Calcif Tissue Int. 2016; 98:165-71.

4. Gao K, Zhu W, Liu W, Ma D, Li H, Yu W, et al. Diagnostic value of the blood monocyte-lymphocyte ratio in knee osteoarthritis. J Int Med Res. 2019;

#### 47(9):4413-21.

5. Bredella MA. Sex Differences in Body Composition. Adv Exp Med Biol. 2017; 1043:9-27.

6. Siervo M, Prado CM, Mire E, Broyles S, Wells JC, Heymsfield S, et al. Body composition indices of a load-capacity model: gender- and BMI-specific reference curves. Public Health Nutr. 2015; 18(7):1245-54.

7. Baumgartner RN, Koehler KM, Gallagher D, Romero L, Heymsfield SB, Ross RR, et al. Epidemiology of sarcopenia among the elderly in New Mexico. Am J Epidemiol. 1998; 147(8):755-63.

8. Litwic A, Edwards MH, Dennison EM, Cooper C: Epidemiology and burden of osteoarthritis. Br Med Bull. 2013; 105:185-99.

9. Lemos T, Gallagher D: Current body composition measurement techniques. Curr Opin Endocrinol Diabetes Obes. 2017; 24(5):310-4.

10. Johnson Stoklossa CA, Sharma AM, Forhan M, Siervo M, Padwal RS, Prado CM. Prevalence of Sarcopenic Obesity in Adults with Class II/III Obesity Using Different Diagnostic Criteria. J Nutr Metab. 2017; 2017;7307618.

11. Ritchie JD, Miller CK, Smiciklas-Wright H. Tanita foot-to-foot bioelectrical impedance analysis system validated in older adults. J Am Diet Assoc. 2005; 105(10):1617-9.

12. Lee JY, Han K, McAlindon TE, Park YG, Park SH. Lower leg muscle mass relates to knee pain in patients with knee osteoarthritis. Int J Rheum Dis. 2018; 21(1):126-33.

13. Abbate LM, Stevens J, Schwartz TA, Renner JB, Helmick CG, Jordan JM: Anthropometric measures, body composition, body fat distribution, and knee osteoarthritis in women. Obesity. 2006; 14(7):1274-81.

14. Sowers MF, Yosef M, Jamadar D, Jacobson J, Karvonen-Gutierrez C, Jaffe M. BMI vs. body composition and radiographically defined osteoarthritis of the knee in women: a 4-year follow-up study. Osteoarthritis Cartilage. 2008; 16(3):367-72. 15. Grotle M, Hagen KB, Natvig B, Dahl FA, Kvien TK: Obesity and osteoarthritis in knee, hip and/or hand: an epidemiological study in the general population with 10 years follow-up. BMC Musculoskelet Disord. 2008; 9:132.

16. Thijssen E, van Caam A, van der Kraan PM: Obesity and osteoarthritis, more than just wear and tear: pivotal roles for inflamed adipose tissue and dyslipidaemia in obesity-induced osteoarthritis. Rheumatology (Oxford). 2015; 54(4):588-600.

17. Farnaghi S, Crawford R, Xiao Y, Prasadam I: Cholesterol metabolism in pathogenesis of osteoarthritis disease. Int J Rheum Dis. 2017; 20(2):131-40.

18. Klop B, Elte JW, Cabezas MC. Dyslipidemia in obesity: mechanisms and potential targets. Nutrients. 2013; 5(4):1218-40.

19. Navab M, Ananthramaiah GM, Reddy ST, Van Lenten BJ, Ansell BJ, Hama S, et al.: The double jeopardy of HDL. Ann Med. 2005; 37(3):173-8.

20. Charles-Lozoya S, Treviño-Báez JD, Ramos-Rivera JA, Rangel-Flores JM, Tamez-Montes JC, Brizuela-Ventura JM. Metabolic syndrome and other factors associated to gonarthrosis. Gac Med Mex. 2017; 153(7):701-5.

21. Feingold KR, Grunfeld C: Effect of inflammation on HDL structure and function. Curr Opin Lipidol. 2016; 27(5):521-30.

22. Welty FK: How do elevated triglycerides and low HDL-cholesterol affect inflammation and atherothrombosis? Curr Cardiol Rep. 2013; 15(9):400.

#### How to cite this article:

Gökhan Peker, Dila Mete Peker, Rahman Köseoğlu, Orkun Gül. Do body composition, hemogram and lipids differ between obese women with and without gonarthrosis? Ann Clin Anal Med 2023;14(5):399-403

This study was approved by the Ethics Committee of University of Health Sciences, Trabzon Kanuni Education and Research Hospital (Date: 2020-12-23, No: 2020/84)